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# Modelling and Validation of Treatment Tables for Severe Decompression Accidents

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## INTRODUCTION

This paper addresses the question of suitable treatment of dysbarism following a severe decompression accident during the use of self-contained breathing apparatus such as the Canadian Underwater Minecountermeasures Apparatus (CUMA) using 1 ata oxygen in helium to a maximum depth of 81 metres. The work involved a dual approach; a theoretical analysis of the problem followed by experimental work designed to follow up specific aspects arising from the theoretical analysis.

## THE MATHEMATICAL MODEL

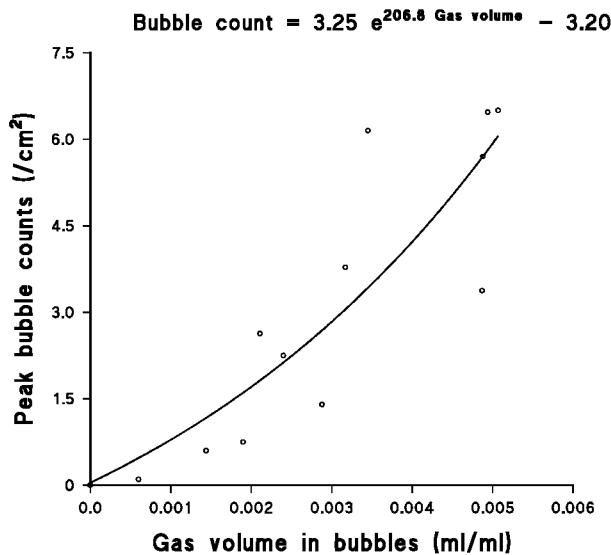
The physiological model of decompression is based on the eight compartment model of gas dynamics described in Mapleson (1963) combined with the model of bubble dynamics described by Van Liew and Burkard (1993). Table 1 lists the compartments together with the time constants which are the factor governing gas uptake.

**TABLE 1**  
**Characteristics of each compartment. Time constant in minutes.**

Compartment	Tissues	Time constant
1	Adrenals, kidneys, thyroid	0.86
2	Heart, brain grey matter	1.87
3	Liver plus portal system, other small glands and organs	3.07
4	Brain white matter	5.31
5	Red marrow	12.25
6	Muscle and skin	50.62
7	Nonfat subcutaneous	69.14
8	Fatty marrow and fat	nitrogen helium
		211.3 78.3

The complete hyperbaric exposure is simulated by making iterative calculations using appropriate time increments. The output for each compartment includes the total volume of inert gas which forms into bubbles; the partial pressures of the inert gases in the tissue, the venous blood and the bubble; the change in bubble radius from an assumed initial size usually taken as 2 mm. These are calculated for each time interval. Venous blood is assumed throughout to be in equilibrium with the tissue which it drains. Gas exchange at the lungs is assumed to be complete on each passage of blood through the lungs. In addition to calculating these parameters for each tissue, weighted means of each are used to calculate the values for central mixed venous blood. This is required for comparison of predicted bubbles with precordial bubble counting.

The conversion from the volume of gas carried as bubbles in the mixed venous blood to predicted pulmonary artery bubble counts is made using the relationship derived from experimental hyperbaric exposures and is shown in figure 1. The bubble counting technique is described in Eftedal et al (1993). The experimental data contributing to the points in figure 1 include over 100 experiments using 14 different types of hyperbaric exposure. The average bubble counts for each series of experiments range from zero to over 6 bubbles/cm<sup>2</sup>, the most severe exposure included animals which died during or shortly after decompression. This relationship has now been used for comparison of model prediction with pulmonary artery bubble counts or grades for many kinds of hyperbaric exposures from a wide range of sources. The relationship between bubble counts and Doppler K-M grades has been taken as that reported by Eftedal et al (1998). Although there are several assumptions implicit in making these conversions the model has performed well. It has also been used to help design decompression trials to give a selected average bubble count (Flook 1999). The work described here represents the most severe test so far in that the model was used to predict both bubble counts at the end of a primary dive and the fate of the bubbles during and following the use of decompression treatment tables; predictions which were then tested in experimental conditions.



**Figure 1** The relationship between average precordial bubble counts in experimental hyperbaric exposures and predicted volume of gas carried as bubbles in the mixed venous blood. See text for details.

## THE THEORETICAL STUDY

The theoretical study focused on 81 metres as the depth of the primary exposure with the most severe exposure being 20 minutes at depth followed by uncontrolled decompression at 10 metres/minute. The start of a treatment recompression was preceded by a surface interval of either 10 minutes or 60 minutes breathing either air or oxygen. Ten minutes was considered the minimum time in which the diver could be recovered and

moved to the treatment chamber. Five treatment tables were considered USN6, USN6A on either air or heliox, RN67 and ECO7A.

The main conclusions from that study were that:

even after only 2 minutes at 81 metres an uncontrolled ascent might be expected to give Doppler grade IV or higher in the average diver;

after a 20 minutes exposure the predicted volume of gas in bubbles exceeded the highest bubble count on figure 1 and exceeded any existing scale of measurement;

recompression on any treatment table resulted in removal of the bubbles;

all treatment tables studied allowed sufficient time for subsequent removal of the gas liberated from the bubbles;

treatment tables which used inert gas as part of the breathing mixture generated new bubbles at some stage in the treatment.

The predicted peak volume of gas carried in bubbles in the mixed venous blood after the 2 minute exposure was 0.010ml/ml with air breathing on the surface, 0.006 ml/ml with oxygen breathing on the surface. After the 20 minute exposure the corresponding numbers were 0.021 ml/ml and 0.015 ml/ml. Given that 20 minutes at 81 metres is an allowed CUMA exposure that was chosen for the experimental study. It was not unreasonable to doubt the model predictions at this stage in the work and to assume that it had over-estimated the amount of gas in bubbles.

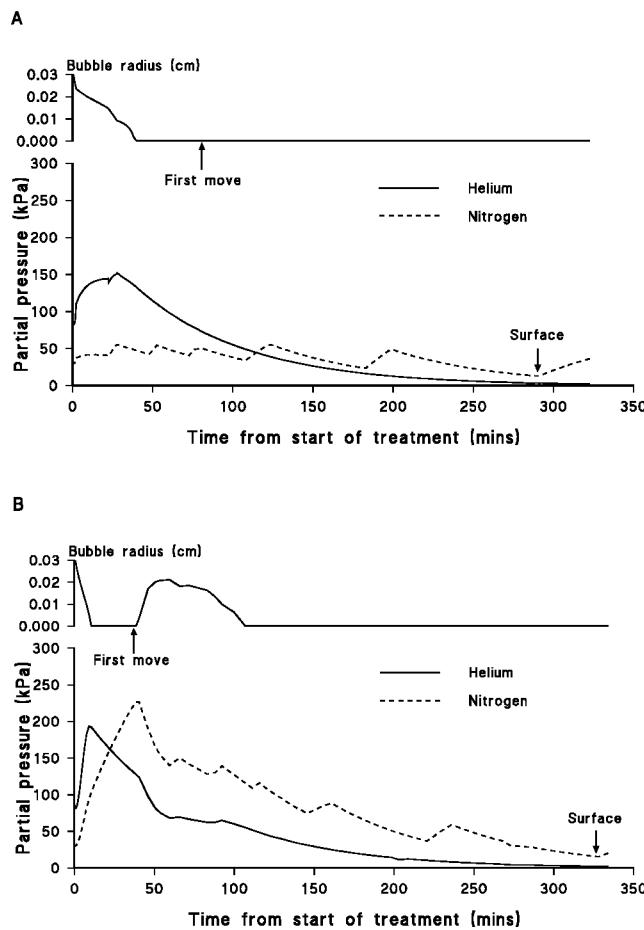
Treatment tables USN6 and USN6A using air are readily provided on site during mine counter measure operations. The experimental work was planned using these for treatment after a 10 minute surface interval during which either air or oxygen was used as the breathing gas.

**Table 2**  
**CUMA81 20 minute blow-up**  
**Time (minutes) to disappearance of bubbles following treatment by USN6 or USN6A**

<b>Compartment</b>	<b>USN6</b>		<b>USN6A</b>	
	<b>Oxygen</b>	<b>Air</b>	<b>Oxygen</b>	<b>Air</b>
<b>1</b>		4.9		10.6
<b>2</b>	3.4	8.9	4.1/40.2	11.6/42.1
<b>3</b>	7.4	9.4	11.1/44.1	22.9/44.1
<b>4</b>	18.4	13.9	15.9/48.1	31.4/48.1
<b>5</b>	43.4	39.9	33.4/62.1	62.6
<b>6</b>	25.4	26.9	6.0/47.1	6.5/88.9
<b>7</b>	31.3	38.9	10.1/99.4	10.6/103.9
<b>8</b>	1.45	N/A	1.4	N/A

Table 2 shows the predicted time to disappearance of bubbles for treatments started after a 10 minute surface interval breathing either oxygen or air. Where 2 numbers are shown bubbles, having disappeared after the recompression, are predicted to reappear on the first move. This is predicted to happen only on USN6A.

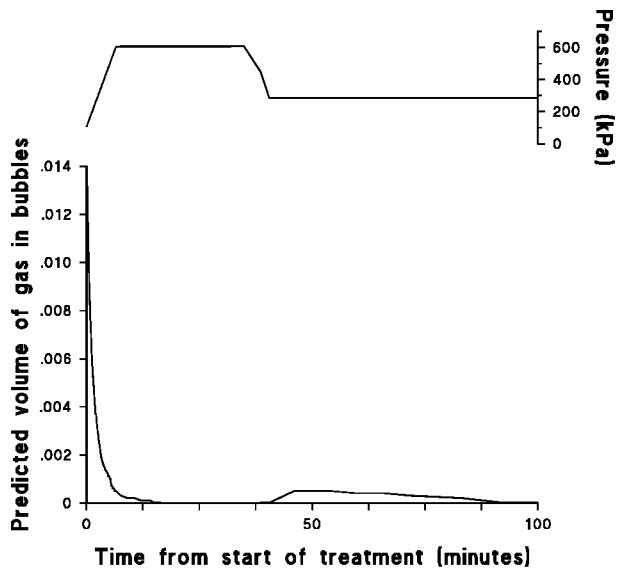
Figure 2 shows the predicted bubble size for compartment 7 during USN6 (figure 2A) and USN6A (figure 2B) following a 20 minutes CUMA 81 with a 10 minutes surface interval breathing air. Two things are of interest; the greater pressure used for the first stage of USN6A results in much faster compression of bubbles, and in this tissue bubbles are predicted to reform on the move from the maximum depth during the USN6A but not during the move from maximum depth on USN6.



**Figure 2** Showing the predicted reformation of bubbles in a tissue following the first move on USN6A and the absence of reformed bubbles following the first move during USN6.

Figure 3 shows the predicted volume of gas in bubbles for the mixed venous blood during the first 100 minutes of treatment by USN6A. This shows quite clearly that bubbles should be detected after the move from maximum depth, though the free gas volume at that time is considerably less than that at the start of treatment.

Thus the experiments were designed to answer the main question; are either of these treatments suitable for the treatment of bubbles following this severe primary exposure? The secondary questions to be answered are; are bubble numbers after the uncontrolled decompression really as high as the model predicts and can bubbles reform after the first move on USN6A?



**Figure 3** The predicted gas in bubbles in the mixed venous blood during the first 100 minutes of USN6A.

### THE EXPERIMENTAL STUDY

The experiments were carried out in the laboratories of SINTEF Unimed in Norway with anaesthetized, spontaneously breathing pigs as the experimental model. The primary exposure was taken as 20 minutes at 81 metres. Ten animals breathed air during the ten minute surface interval, ten animals breathed oxygen.

Details of the normal laboratory routines are described in Reinertsen et al 1998. All experiments were approved by the Norwegian Committee for Animal Experiments. Chamber pressure, rate of change of pressure, breathing gases and gas switches were all controlled automatically using a system designed in the laboratory, (Kleven 1991). Bubble counts were recorded at one minute intervals; inspired and mixed expired gases were recorded at half minute intervals as was inspiratory flow, by Fleisch V pneumotachograph. No blood pressures were recorded as the decision was taken to minimise surgical intervention because of the possibility that such intervention and insertion of catheters could perhaps influence bubble numbers. The level of anaesthesia and general well being of the animals was monitored by blood gas analysis carried out on venous blood drawn from the ear. This was done during the stabilisation hour which preceded the primary compression and then not again until the first move of the treatment table was completed and after confirmation that bubbles had or had not reformed as a result of that move.

In addition to transoesophageal echocardiographic bubble detection (at 7.5 MHz) a femoral vein was exposed and a 10 MHz ultrasonic probe placed to allow detection of bubbles which must mainly derive from the large muscles of the leg.

### THE EXPERIMENTAL RESULTS

Having minimised the extent of surgical intervention there are few physiological parameters available to indicate the well being of the animals before decompression. Table 3 shows the average mixed expired oxygen and carbon dioxide values for the two series during the last five minutes at 81 metres, before the primary decompression. Average inspired oxygen was 11.1%.

**TABLE 3**  
**Expired gases prior to primary decompression**

	<b>Expired oxygen (%)</b>	<b>Expired carbon dioxide (%)</b>
<b>Air on surface interval</b>	$10.5 \pm 0.26$	$0.25 \pm 0.07$
<b>Oxygen on surface interval</b>	$10.46 \pm 0.11$	$0.25 \pm 0.03$

The two groups are essentially the same, the variance within each group is low and the gas values are as expected in healthy animals.

The experimental results following decompression are difficult to present as a group not least because the model prediction proved to be correct; an uncontrolled decompression from 20 minutes CUMA81 is a very severe, often fatal, experience. Even so some animals had few detectable bubbles during the ten minute surface interval. This variability is a common feature not only in animal experiments but also in experimental and operational exposures in humans.

Table 4 gives details of the outcome for the animals which breathed air during the surface interval. The number in brackets indicates the time of maximum bubble counts, decompression is completed at 84 minutes. The experiments marked \* do not record the true maximum bubble counts. These animals died very quickly after surfacing at a time when bubbles counts were increasing rapidly. Not only do we fail to record the maximum because of the one minute interval between recordings but also, once the animal goes into circulatory failure, the number of bubbles appearing under the probe is determined by the blood flow. This means that there is not a meaningful value for the average maximum bubbles but the average of the values listed in Table 4 is 9.4 bubbles/cm<sup>2</sup>, very much higher than the highest values shown in figure 1.

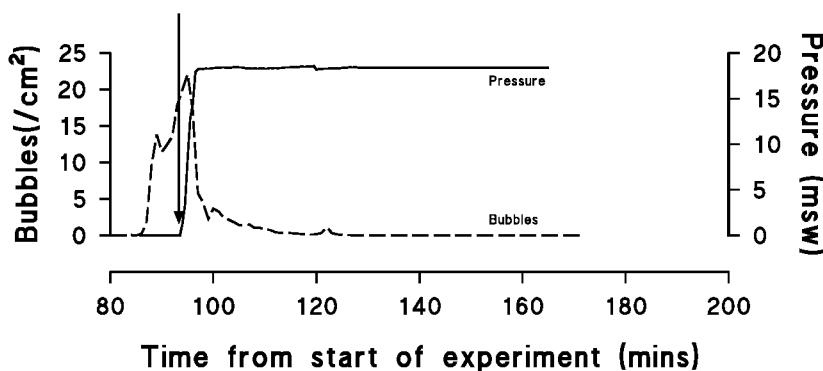
**Table 4**  
**Maximum recorded bubbles counts in pulmonary artery and femoral vein**

	<b>PA Bubbles (/cm<sup>2</sup>)</b>	<b>Femoral bubbles</b>	<b>Outcome</b>
<b>Expt 1</b>	0	0	No bubbles
<b>Expt 5*</b>	5.74	1203	Died before treatment
<b>Expt 9*</b>	5.57	474	Died before treatment
<b>Expt 10</b>	8.54 (94)	436	Completed USN6
<b>Expt 11*</b>	9.35	702	Died before treatment
<b>Expt 12</b>	0.11 (95)	0	Completed USN6
<b>Expt 13</b>	15.17 (89)	596	Died after USN6A compression
<b>Expt 14*</b>	7.41	1818	Died before treatment
<b>Expt 15</b>	22.56 (91)	1821	Died during USN6A
<b>Expt 16</b>	19.16 (94)	701	Died after USN6A compression

Table 5 shows the same information for the animals which breathed oxygen during the surface interval. In this series all animals survived to be recompressed and therefore the average maximum bubble counts is a more meaningful number though should still be interpreted with caution as, for example, animal 2/8 was recorded in the laboratory log as being in circulatory failure, very high heart rate reduced blood flow, at the time the maximum value was recorded. The bubbles counts are referred to as "maximum" rather than "peak" because in 2 animals this value was recorded after the treatment recompression had actually started. Figure 4 shows the time course of bubble numbers for one experiment with the arrow marking the start of recompression. Bubble numbers may not have reached a true peak before recompression started. Once again there is a very wide range of maximum counts in these experiments.

**Table 5**  
**Maximum recorded bubble count in pulmonary artery and femoral vein**

	PA Bubbles (/cm <sup>2</sup> )	Femoral bubbles	Outcome
<b>Expt 7</b>	5.6 (94)	0	Completed USN6A
<b>Expt 8</b>	0.07 (99)	386	Completed USN6
<b>Expt 2/1</b>	20.52 (96)	1175	Died during USN6
<b>Expt 2/2</b>	0.04 (86)	0.146	Completed USN6
<b>Expt 2/3</b>	16.01 (86)	1964	Died after USN6A compression
<b>Expt 2/4</b>	22.1 (85)	1928	Died during USN6
<b>Expt 2/5</b>	21.07 (92)	1642	Died after USN6A compression
<b>Expt 2/6</b>	16.74 (94)	1694	Completed USN6
<b>Expt 2/7</b>	0.04 (86)	0	Completed USN6A
<b>Expt 2/8</b>	20.94 (89)	1825	Died after USN6A compression
<b>Average</b>	<b>12.3 ± 9.7</b>		



**Figure 4** Time course of bubble numbers relative to pressure profile

The failure to record true peak bubble counts means that it is impossible to evaluate the effect of oxygen breathing during the surface interval though it does seem that the oxygen allowed more animals to survive to begin treatment.

Table 6 lists the experiments in which the animals survived to, at least, beyond the first move of the treatment decompression. Both air breathing and oxygen breathing experiments are included. Time to resolution of bubbles is measured from the start of the recompression and relates to the bubbles which resulted from the primary exposure.

**TABLE 6**  
**Bubble history for animals which survived beyond first move of treatment**

	PA Bubbles (/cm <sup>2</sup> )	Time to resolution of bubbles	Later bubbles	Table
<b>Expt 10</b>	6.5	43.5	None	USN6
<b>Expt 12</b>	0.11	11.5	None	USN6
<b>Expt 15</b>	22.6	26.5	0.77	USN6A
<b>Expt 7</b>	5.6	31.5	0.03	USN6A
<b>Expt 8</b>	0.07	7.5	None	USN6
<b>Expt 2/2</b>	0.04	before treatment	None	USN6
<b>Expt 2/6</b>	16.74	54	None	USN6
<b>Expt 2/7</b>	0.04	before treatment	None	USN6A

The wide range of bubble counts from the primary exposure, and the small number of animals which completed treatment to beyond the first move, make it difficult to quantify the results. However some tentative conclusions can be drawn. All animals in Table 6 except #15 completed the treatment. None showed any evidence of reformation of bubbles at the end of the treatment. Both USN6 and USN6A are shown to get rid of the bubbles and to do so well before the end of the treatment. These treatments continue long enough for the liberated gas to be cleared from the body. The most useful experiment from this point of view is #2/6 in which bubbles from the primary dive were certainly high enough to come within the range in which animals frequently die. This is the strongest evidence that USN6 is an adequate treatment table for this kind of accident.

With the exception of #15, which died during treatment and therefore could have been in circulatory failure, there is a relationship between primary bubble count and time to resolution of bubbles with some evidence that USN6 takes longer than USN6A to resolve the bubbles.

Of the three animals which started USN6A two had a recurrence of bubbles after the first move. None of the four which had USN6 had a recurrence of bubbles.

## CONCLUSIONS

Although it would have been preferable to have a more homogenous set of experimental results, and more definitive conclusions, the experimental results are much as predicted. The first conclusion is that an uncontrolled decompression from 20 minutes CUMA81 is more likely than not to be a fatal experience. Secondly both USN6 and USN6A can resolve the bubbles and appear to do so with sufficient treatment time left to clear the liberated gas. The highest bubble count for which USN6 was successful was in experiment 2/6, a peak bubble count of  $16.7/\text{cm}^2$ , well in excess of K-M score IV. Thirdly there is some evidence that the presence of inert gas in the breathing mixture at the maximum depth on USN6A can cause bubbles on the first move, whereas there is no evidence of reformation of bubbles on USN6.

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